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SOME MEDICAL ASPECTS OF THE DISEASES OF THE GALL-BLADDER AND GALL-DUCTS

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INTRODUCTION

It seems almost a work of supererogation to discuss the subject that we are to consider to-day. In recent months so many and valuable have been the papers published that it has seemed as though the very words were taken out of the writer's mouth, so that what is to be said to-day smacks to him of repetition.

Comparison with Appendicitis.—It need not, however, be vain repetition. The era we are about to enter upon bodes for good. In the treatment of gall-bladder infections it looks as if we would go through the cycles of activity and inactivity, as in the early periods of our knowledge of appendicitis.

Primary Disease to be Treated.—We trust that this contribution will further the progress which the writer sincerely believes is the true one, to the end that primary disease of the gall-bladder and gall-ducts will be accurately diagnosed, and precisely and hence thoroughly treated, so that secondary disease will be relatively rare. Local causes, such as displacements and conditions which give rise to external pressure, must be removed. As forced upon us now, it is secondary manifestations that we have to contend against, manifestations which imply complications so difficult to manage as to render the mortality of alarming proportions.

Secondary Disease Fatal.—The opprobrium of the medical and surgical treatment of gall-duct and gall-bladder affections arises because the death-rate in secondary affections is so high. Let us see to it that the primary ailments are cared for, blocking thereby the oncoming of secondary conditions.

Prognosis.—If primary processes are recognized in the future, cholelithiasis with its enormous train of abnormal states, obstructions and dilatations, will be prevented. Modern treatment of these affections will result in success in the highest aim of our endeavor, prevention. Fifty years from now the many secondary phenomena will be as rare as those which are secondary to diseased conditions now removed early, such as cataract, appendicitis, vesical calculus, and a host of other conditions. Students of that period will marvel at the reports of the cases of the present.

Morbid Anatomy of the Future.—It is interesting to picture what a blank there may be in the morbid anatomy of biliary affections fifty years hence. The present day pathologist should make haste to fit out his anatomical museum to full completion in respect to biliary pathology. We can point out that notwithstanding the extraordinary multiplicity of primary and secondary lesions, our museums contain but few illustrations of these processes.

Museum Specimens Few.—A search of the museums of Philadelphia disclosed less than twenty prepared specimens, excluding gall-stones, which a student could consult.

Surgeon's Need.—This was not surprising, for the writer has seen surgeon after surgeon operate on biliary disease when they had no conception of the anatomical complications that arise, as in a case of prolonged cholecystitis and cholelithiasis, and they readily became involved, indeed lost, in a morbid anatomy quagmire as effectually as if thrown into a yawning abyss in the dismal swamp. The first precept the writer would urge is, Surgeons should know morbid anatomy.

Terminal Infections.—Before leaving the field of morbid anatomy, a word further. My own studies have impressed me with the great frequency with which disease of the gall-bladder and gall-ducts lead to the termination of life. In many, many cases, put down as senility, in cases of chronic disease, as tuberculosis, nephritis, cardiac disease, death has not been due to these affections, but to terminal infections, the source of which was in the biliary appa-

ratus. Terminal infections will therefore be prevented by the removal of primary foci of disease.

GENERAL SUMMARY

Morbid States.

A. The primary morbid states of the gall-bladder and gall-ducts are:

- Inflammations { catarrhal (rare). / infectious.
- Morbid growths (primary).
- Parasitic invasions.

B. The secondary morbid states are:

(1) Causes outside of the gall-bladder and gall-ducts.

- Inflammations { catarrhal { secondary to heart, lung, / infectious { or abdominal disease.
- Morbid growths.
- Obstructions { displacements and deformities. / diseases outside of the ducts.

(2) Causes within the gall-bladder and gall-ducts.

- Inflammations (including ulceration and perforation).
  - Because of { displacements and deformities. / morbid growths.
- Foreign bodies { parasites. / gall-stones.
- Obstructions, because of
  - Inflammations,
  - Morbid growths,
  - Foreign bodies.
- Enlargements, because of
  - Inflammations,
  - Morbid growths,
  - Foreign bodies,
  - Obstructions.

Unfortunately, the states which clinicians see are in the large proportion of cases secondary, and belong to

(1) Local disease, as indicated above.

(2) Diseases in remote parts.

PRIMARY MORBID STATES

INFLAMMATION OF THE GALL-DUCTS. CATARRHAL CHOLANGITIS

Causes.—The disease is secondary to gastroduodenal catarrh, to pressure, to local spreading infections. Other cases of catarrhal jaundice are found to be primarily infectious.

The *symptoms* and signs are well known. The age of the patient, the presence of a cause, and the clinical course make up the picture. When long continued, catarrhal inflammation may resemble obstruction due to other primary or secondary processes.

The *diagnosis* in cases of so-called catarrhal jaundice continuing more than six weeks should be revised if the erythrocytes fall in number, the hemoglobin drops, the spleen enlarges, and there is loss of weight. The true nature of the disease can be determined by the antecedents or by accompaniments, which by this time may be more prominent, such as the enlarged glands of syphilis or tuberculosis. Organic diseases, cirrhosis, cancer of the liver, infections, as Weil's disease, must be excluded. Many so-called catarrhs are the result of typhoid, pneumococcus, or other infections.

Treatment. — Medicinal. — Hydrotherapy, including an abundance of water internally; enemas of cold water (Krull); compresses; baths. Proper diet (Carlsbad at home). Clothing. Exercise. Rest. Alkalies; iron, if anemic. Ammonium chlorid.

Surgical.—Symptomatic operative interference is compulsory, if the obstruction is complete, jaundice prolonged, the patient toxic, and the liver or the spleen enlarged.

SUPPURATIVE CHOLANGITIS

Suppurative cholangitis simulates pyelophlebitis, abscess of the liver, syphilis of the liver, rare cases of cancer, and Weil's disease.

Diagnosis is based on: (1) *Clinical Course.* — The onset is gradual. There is infection of the ducts or the gall-bladder; foreign bodies, as gall-stones, are present, or there was a previous general infection.

(2) *Objective Diagnosis.*—Icterus may or may not be present. The fever is characteristic (hectic type). Toxic symptoms from sepsis of jaundice occur.

(3) *Physical Diagnosis.*—The liver is enlarged moderately and tender. There is a tender area in the region of the twelfth dorsal vertebra, from 2 to 3 cm. from the middle line (Boas).

(4) *Laboratory Diagnosis.*—Leukoeytosis. Serum reaction to determine eause. Blood examination to exelude malaria.

Differential Diagnosis (see Infeetions).—Pyelophlebitis: absenee of eause for infeetion in the portal area. Amebic abseess: elinical antecedents, physical diagnosis, and absenee of leukoeytosis. Malaria: blood examinations. Simple eholelithiasis: no leukoeytosis (see Ball-valve Caleulus).

Treatment eonsists of drainage. Patience and waiting are of doubtful value.

CHOLECYSTITIS

Choleeystitis simulates hyperemia and infections of the liver, sub-diaphragmatic abscess (see Mason's and Osler's papers), diaphragmatie pleurisy, pneumonia, panereatic affeetions, perforations of uleer of the stomach or duodenum, intestinal obstruetions, uremia, and endoearditis.

Diagnosis is based on: (1) *Clinical Course.*—Primary eholecystitis follows acute gastroduodenitis, and general infeetions. Secondary eholeeystitis follows infeetions indueed by foreign bodies and obstruetion. It oeeurs at any age and in either sex; habits of but little eonsequenee. Anteeedents mentioned above. The onset varies; is often fulminating, but may be mild and gradual or severe (see report of eases, pages 22 to 29).

(2) *Subjective Diagnosis.*—Pain from spasm or tension, tenderness, nausea, and vomiting.

(3) *Objective Diagnosis.*—Fever, mild to severe. Remittent or intermittent. Chills in fulminating type. Jaundiee not present.

(4) *Physical Diagnosis.*—A tumor at the end of the ninth rib. Has the eharaeteristies of a swollen gall-bladder; movable with respiration unless inhibited by pain. Spasm of reetus musele.

(5) *Laboratory Diagnosis.*—Leukoeytosis in all infeetions except those due to typhoid fever. Present in 50 per cent. of the latter. Serum diagnosis is important to determine the nature of the infeetion.

CHOLECYSTITIS IN TYPHOID FEVER

A loeal infection of the gall-bladder in typhoid fever may develop in the course of the disease—during the third or fourth week, or in the period of convaleseence. All grades are seen. It may or may not be aeeompanied by eholangitis. If it oeeurs in

the course of the disease its onset may be marked by an increase in the temperature, varying with the intensity of the local infection. In mild forms the fever may become a little higher than the continued range that has preceded. In severe forms it may become remittent or intermittent, and chills are not uncommon.

Nausea and vomiting are common in the beginning and may continue for several days. Pain in the region of the gall-bladder, localized tenderness, and rigidity of the right rectus muscle develop with the fever.

Fulminating cases, simulating perforation of the bowel, may occur during the course of the disease.

The mode of onset and association with cholangitis is illustrated by Case II. The symptoms are very characteristic. Pain is often so extreme as to simulate gall-stones or lead to collapse. It is usually in the situation described, but may be referred to the epigastrium. The pain may seem to be general at first and even be marked away from the gall-bladder, as in the right iliac fossa. Often the pain is diffused during the first 24 hours and attended by general rigidity and distention. Frequently the local symptoms are overlooked, and I am convinced that many cases of so-called relapse in typhoid fever are due to mild cholecystitis.

Leukocytosis need not be present. The data is quite insufficient concerning this point. In one of my cases it was absent, although following the law of leukocyte increase, such absence might have been due to the extreme infection.

Cholecystitis during convalescence from typhoid fever is not unusual. Contrary to Da Costa's experience, I have seen it quite as frequently at this period as at any other. Here, too, the cases may be mild, fulminating, and perforating. Case III will illustrate the onset and course of a fulminating attack.

Briefly, the case (III) seen with Dr. Rehfuss was one of typhoid fever of long duration in a woman, 56 years of age. Five days after the temperature was normal she was seized suddenly with severe diffuse abdominal pain. The greatest tenderness seemed to be confined to the right iliac fossa. She had a chill coincidentally, followed by fever and much prostration. I saw her two days after the onset of pain. Although no defined tumor was present the pain, rigidity, and tenderness were confined to the right upper quadrant; vomiting persisted, tympany was marked. Three hours after my

visit Dr. Edward Martin operated. In the interval the signs of inflammation extended over the upper abdomen. The gall-bladder was much enlarged and attached to neighboring organs by adhesions. It contained mucus and pus, both bile stained. There were no calculi, and the ducts seemed free. Operation was followed by immediate relief and an uninterrupted convalescence (see charts).

Case IV was one of onset more gradual after the typhoid process was completed. It is interesting to note that tenderness was absent, leukocytosis was absent, while chills and fever were dominant features. The chart herewith exhibited shows the course of the fever. The occurrence of death from acute hemorrhagic pancreatitis is most interesting in the light of recent researches.[1]

The onset in the post-febrile period takes place in the first or second week, when we are about to place the patient on solid food. In consequence, the change of diet is held responsible for the pain, tympany, and fever.

DIFFICULTY IN RECOGNIZING SECONDARY CHOLECYSTITIS

Cholecystitis occurring in a subject who has gall-stones or a displaced liver differs from primary cholecystitis in local rather than general symptoms. The altered position of the gall-bladder changes the location of the pain and tumor. In a case operated on by Dr. Harte the tumor was below the transverse umbilical line, and in one operated on by Dr. Price the tumor was in the median line and the gall-bladder had emptied its contents into the lesser peritoneal cavity. In the first case the tumor was continuous with the liver dulness, in the second it was not. It is scarcely necessary to say that the antecedent history of cholelithiasis aided in the diagnosis.

DIFFERENTIAL DIAGNOSIS

My experiences of the past year have led me to believe that cholecystitis can easily be recognized, the difficulty arising in distinguishing some forms of it from appendicitis being greatest, and yet these are cases of secondary cholecystitis and therefore more obscure because due to displaced gall-bladders. Primary cholecystitis can be recognized by the clinical course, the physical signs, and

[1] Since this paper was read, Moynihan has reported a similar case in the Brit. Med. Jour., 1903, i, 1350. These are the first cases of this character reported.

the associate symptoms, together with the results of laboratory diagnosis.

In the distinction between the *catarrhal* and *suppurative* varieties we are deciding in part between mild and severe forms of infection. The degree of the infection is estimated by the severity of the local symptoms and the intensity of the toxemia as indicated by the fever, the cardiovascular and cerebral symptoms, and the leukocytosis. It is to be remembered that an intense alarming inflammation may arise without pus formation, as in Case I. A clear fluid was removed from the gall-bladder.

The following diseases are to be considered in the differential diagnosis:

(1) *Congestion of the Liver.*—In cases of acute infectious endocarditis with cardiac dilatation, the left lobe of the liver often enlarges and is the seat of pain and great tenderness. This is accompanied by jaundice. An enlarged gall-bladder may be simulated, and as the general symptoms of infection prevail, that organ may be considered the site of primary infection. It is not unusual to have endocarditis without physical signs in the heart. Again, in a case of purulent pericarditis under my care, the distention of the liver capsule was extreme, pain was excessive, and the picture was not unlike that of suppuration of the biliary passages.

(2) *Syphilis of the Liver.*—The history may help, but who does not know how weak such help is in this infection. Antecedent and associate conditions aid us here. The fever is commonly intermittent in syphilis. The paroxysms are at fixed periods in the day, however. Leukocytosis is absent. Justus's sign is not satisfactory. A localized tumor with the characteristics of an enlarged gall-bladder is usually not present. It may, however, exist, although it is not so tender. A female patient of mine, aged 32 years, who had been exposed to the possibility of infection, was seized with pain, tenderness, and swelling in the gall-bladder region. She was told it was "cold" or "gall-stones." Jaundice followed. The local tenderness and tumor continued but the jaundice disappeared. The mass simulated cancer of the pylorus. A moderate fever prevailed. Specific treatment dissipated the tumor, and all the symptoms vanished with it. There has been no recurrence in twelve years. A rapidly growing localized gumma attended by fever and sweats may simulate gall-bladder infection. It has been my experience to find a lymphocytosis more common in syphilis than in other infections.

(3) *Multiple Abscess of the Liver or Pyelophlebitis.—Portal Pyemia.*—The differential diagnosis is sometimes difficult or impossible. The antecedent history of abdominal infection is necessary to establish the diagnosis. The liver is large in multiple abscess; in cholecystitis the gall-bladder is large. Pain is not marked in multiple abscess. Ascites, enlarged spleen, hematemesis, and diarrhea are more common in portal obstruction.

(4) *Abscess of the liver* occurring in amebic dysentery is recognized by the history, by physical signs posteriorly rather than anteriorly, and often thoracic rather than abdominal, by the frequent absence of leukocytosis, the rarity of fever whose type is more or less slow, and by the presence of uniform enlargement or enlargement in one direction of the liver. The spleen is more likely to be enlarged in abscess than in non-typhoid cholecystitis. Abscess of the liver is neither as painful nor as acute an infection, and hence not as intense an infection as cholecystitis.

(5) *Secondary Cholecystitis.*—It is to be remembered that we are considering only the differentiation of primary cholecystitis and hepatic affections. Secondary cholecystitis and cholangitis have the history of the primary cause.

(6) *Subdiaphragmatic Abscess.*—History, physical signs, and exploratory puncture avail (see papers of Mason, Osler, and others). It manifests itself posteriorly, and signs are abdominal as well as thoracic. Difficulty of diagnosis is not great in primary cholecystitis.

(7) *Diaphragmatic Pleurisy.*—Diagnosis at times is difficult. Exposure to cold is a feature in pleurisy. The following incidents in a case under my care illustrated certain points in diagnosis: The patient, who was robust, although rheumatic, was operated upon for hemorrhoids. Three days later, after taking a cold bath, symptoms of diaphragmatic pleurisy, but suggestive of gall-bladder infection, developed. There were chill and fever, and rheumatism in other muscles, but there was no leukocytosis. The diagnosis of pleurisy was made.

(8) *Pneumonia.*—It is only necessary to call attention to the likelihood of confusing the infections in some cases. In pneumococcus infection with gastric and abdominal symptoms dominant, the pulse-respiration ratio and the expiratory grunt alone may suggest the lesion, especially in children. In those in whom jaundice occurs early the confusion may be increased.

(9) *Pancreatic Diseases.*—These have been so exhaustively considered at yesterday's session of this Congress that it is not necessary to take them up on this occasion.

(10) *Perforation of Gastric and Duodenal Ulcer.*—The diagnosis is sometimes difficult, but the marked difference in the previous history is most helpful. Without such carefully worked out history diagnosis may be impossible.

(11) *Intestinal Obstruction.*—This condition does not simulate hepatic and biliary disorders as frequently as it does pancreatic lesions. Time forbids an extensive review.

(12) *Uremia.*—Like hysteria, uremia can simulate almost any other condition. In that form in which abdominal pain and vomiting are most prominent, the surgeon may easily be led astray, particularly as albumin and casts are almost always present in gall-bladder infections.

PRIMARY CANCER OF THE GALL-BLADDER AND GALL-DUCTS

The literature of this affection has been thoroughly studied. The cases collected by the writer, Courvoisier, Sugist, Ames, Kelynack, Rolleston, and others are accessible. It may be said that our knowledge of this condition is fairly definite.

PARASITES

Parasitic invasions of the biliary passages, as by hydatids or round worms, lead to obstruction of the ducts and perhaps enlargement of the gall-bladder, with secondary catarrhal or suppurative cholangitis. A shrewd guess at the diagnosis may be made by exclusion and by the antecedent history of these affections. Otherwise the cases fall under the head of inflammation or of obstruction of the biliary passages.

SECONDARY MORBID STATES

It is not within the province of this paper to consider all the lesions that arise secondarily to primary morbid states. A few will be considered. Thus it is important to consider *displacements* and *deformities* of the liver, as they give rise to many pathological conditions of the ducts or simulate gall-bladder diseases. It is hoped that this study of gross conditions will lead to the study of

less evident states, for the writer fully believes that displaeements are leading etiologieal faetors in biliary affeetions, and that we are ignorant of the relatively normal position of the liver and do not know when the liver is displaeed in minor degrees. The prevention and treatment of minor displacements is obvious.

Morphology.—In regard to gall-stones and displacements it is rare to see cholelithiasis, not of infeetious origin, in persons who are morphologieally of normal type. The patient is too fat or too lean; too small in girth or perhaps too large; presents one of many abnormal types of thorax and abdomen.

DISPLACEMENTS AND DEFORMITIES[1]

A. Displacements.—About 80 cases have been reported. Gall-bladder and gall-duet disease is simulated beeause

(1) Gall-stone eolic is simulated by obstruetion of the duets;

(2) Of obstruetive jaundiee due to kinking of the ducts;

(3) The tumor simulates a gall-bladder tumor.

Diagnosis.—General morphology is suggestive. Clinieal eourse. Females. History of trauma or abdominal disease. Diastasis. Long duration. Reeurrent attacks of pain, transient jaundiee, bilious vomiting. Symptoms of pressure upon other organs. Neurasthenia. Gastro-enteroptosis. Fever absent, exeept in a few eases.

Physical Examination.—(1) Tumor of the size, shape, consistenee, and movability of the liver.

(2) Tympanitie note over the normal area of liver dulness.

Note.—Pereuss in the upright and the reeumbent postures.

Exception.—Anteversion of liver when eonvex surfaee is in contaet with the diaphragm and the anterior abdominal walls—dulness not absent.

(3) Palpation by Glenard's procédé du pouee.

(4) Replaeed by palpation or recumbent posture.

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[1] Literature:

Landau, Die Wanderleber, etc., 1885.

Faure, Thèse de Paris, 1892.

Graham, Trans. Assoc. Amer. Phys., 1895, x, 258. (Résumé of 69 cases.)

Paekard, Univ. Med. Mag., 1897.

Teleky, Centralblatt für die Grenzgebiete der Mediein und Chirurgie, 1901, p. 267.

Steele, Experimental Evidences of Biliary Obstruetion in Floating Liver, Univ. Med. Mag., Dec., 1902.

B. Deformities.—(1) *Floating Lobes.*—Riedel's lobe. Tongue-like process. Gall-bladder tumors simulated by elongated lobe.

Diagnosis.—Clinical course uneventful; or tumor in which attacks of pain and tenderness. Usual occurrence in women; history of abdominal constriction. Attacks of gall-stone colic and cholangitis, a coincidence or rarely a sequence.

Physical Examination.—Tumor movable with respiration, variable size, tongue-like, connection with liver may or may not be demonstrated by percussion. Palpation: movable, sometimes disappears. In rare instances behaves like floating kidney. Percussion: Dull if thick; resonant if thin. Solid, smooth.

Cholecystitis simulated by tumor, which at times is the seat of pain and tenderness. Cholelithiasis is a frequent accompaniment, and may give rise to confusion.

(2) *Corset Liver.*—Gall-bladder tumor simulated by this deformity.

Clinical Course.—Like that of floating lobe; often associated with incidents of hepatic disease, hence may be coincident with cholecystitis, cholangitis, cholelithiasis, gastro-enteroptosis, and carcinoma.

Physical Examination.—Tumor as in floating lobe. If separated from liver, variable in size and shape; consistence of liver; movable to an extreme degree; never in the loin; tender and swollen; always superficial; sometimes the seat of spontaneous pain.

LIMITATION OF OUR KNOWLEDGE IN BILIARY AFFECTIONS

We have much to learn of the pathology and clinical course of these affections.

(1) *Leukocytosis.*—We cannot as yet consider it more than a symptom. The recorded cases are few that give information either by numerical or differential count. My observations go to show that leukocytosis is present in all primary infections except typhoid fever; absent or uncertain in all secondary infections. Its presence therefore points to a primary lesion.

(2) *Hepatic Function.*—We have no clinical method of determining the degree or character of alteration of function in hepatic, much less biliary, disorders. The studies of Edsall, incorporated later, show how little we can rely on the urine for the clinical diag-

nosis of hepatic disorders. Perhaps we need a research hospital for the study of hepatic disease more than of any other organs.

(3) *Displacements* are obscure. We do not know what constitutes a displacement sufficient to predispose if not excite biliary disorder. The questions of morphology and preventive measures are too vast for discussion. It is not dislocations but minor displacements of which we must learn more. What degree of displacement gives rise to impeded circulation of the bile in the channels—interferes with bile drainage?

(4) *Treatment.*—We have now accurate knowledge of the effects of treatment. Do "cures" postpone the evil day? How many persons get well permanently and without secondary states always pending, if not in full blast? Is it worth while waiting for medicinal treatment in primary cases? How many get well in this manner and how many experience temporary cure, even if they get relief? Finally, do surgical measures in acute primary infections prevent secondary states? It seems too soon to answer the latter question. My personal experience supports the knife as the best cure in cholecystitis.

(5) *Morbid Anatomy.*—The young surgeon should see the autopsies of at least 500 cases of old people whereby at least 100 cases of secondary diseases of the gall-bladder and gall-ducts would be studied.

(6) *Operative Measures.*—When should an operation be advised? In catarrhal cholangitis, if chronic, that is, of six weeks' to two months' duration, with no improvement; if the blood is falling, if the time required for blood-clotting increases, and if the spleen and liver are enlarging, operation should be done. In *suppuration* operate in all cases, if no relief to any one symptom in three days; if the liver does not increase, if the fever falls, if the leukocytes fall, if jaundice improves, use expectant treatment. In *cholecystitis* operation perhaps should be performed in every case. Certainly operate in fulminating cases with sepsis and progressive toxemia; leukocytes increasing or stationary. If cultures or serum reaction indicate the infective organism, be guided by its character. A colon infection means operation. The question of operative interference must be decided not alone by laboratory investigation but by clinical sense. If a patient is sick to-day, sicker the next day, and is a little more toxic and septic each day, an operation should be done, in

spite of the absence of leukocytosis. The matter is one of degree of illness, and in each case the clinical acumen of the physician must stand in some service.

Nephritis is not a contraindication to operation. "Bile" and septic nephritis are quickly relieved by the operation.

DEFINITE KNOWLEDGE CONCERNING BILIARY AFFECTIONS

We deem that it can be said that we have fairly definite knowledge concerning diseases of the gall-bladder and gall-ducts. With the limitations already described borne in mind we can say we have a good clinical picture of

(1) Acute catarrhal and suppurative cholangitis.

(2) Cholecystitis.

(3) Primary carcinoma.

(4) Displaced or deformed liver if in excess.

(5) Ball-valve calculus, a symptom group well worked out by Naunyn, Fenger, Osler, and others.

(6) Courvoisier's law.

(7) Points in physical diagnosis described under special diagnosis.

(8) Points in laboratory diagnosis.

SPECIAL DIAGNOSIS OF AFFECTIONS OF THE BILIARY PASSAGES

We must determine if there is present,

A. As primary states,
- Inflammation.
- Morbid growth.
- Parasitic invasion.

B. As secondary states,
- Inflammation and its results.
- Cholelithiasis.
- Obstruction { displacements and deformity. / inflammation. / foreign bodies. / external disease.
- Morbid growth.
- Enlargements.

The recognition of primary or secondary states can only be ob-

tained by the employment of all means of diagnosis at our command.

It may be said that *laboratory* and *physical diagnosis* are of the greater value in *primary* states of the biliary passages; *historical* diagnosis in the *secondary* states. Some points elicited by each method are herein detailed.

Subjective Diagnosis.—The various subjective symptoms and their vagaries are familiar. It is well to recall that the pain of gall-bladder disease is due to inflammation, to distention, to colic or spasm induced by a foreign body (gall-stones). The pain due to inflammation and spasm and simulating hepatic colic must be distinguished from:

(1) Dietl's crises, in floating kidneys, which may be accompanied by jaundice.

(2) Crises occurring in dilated and displaced stomachs, first described by Kussmaul, due to kinking and stenosis of the first and second portions of the duodenum, especially if adhesions have taken place.

(3) Crises due to downward displacement of the liver (36 per cent. of cases—Steele).

(4) Renal calculi.

(5) Pancreatic pain.

Hemorrhagic pancreatitis.

Acute pancreatitis, occasional jaundice.

Pancreatic colic, crisis, and sometimes jaundice.

(6) Gastric ulcer with local peritonitis.

(7) Duodenal ulcer—jaundice in a small proportion of cases.

(8) Gastric neuroses.

(9) Abdominal pain of thoracic disease, as pneumonia, diaphragmatic pleurisy.

(10) Disease of the vertebræ—tuberculous and rhizomelic.

(11) Appendicitis.

(12) Intestinal pain.

Acute ileocolitis.

Lead colic.

Epigastric hernia.

(13) Gastric crises of locomotor ataxia.

(14) Peritoneal pains of toxic origin often occurring in uremia—pain in upper half of abdomen, more or less collapse with nausea and vomiting.

(15) Pain resulting from a forgotten fracture of a rib with callus pinching the nerve.

(16) Aneurism of the aorta.

(17) Obscure alleged neuralgias of the liver.

Objective Diagnosis.—Nothing unusual need be pointed out except as to the temperature. We have striking temperature ranges in hepatic disorder. One type to which Charcot first called attention is intermitting. This type, usually associated with ball-valve calculus, may be confounded with malaria (plasmodium), septicemia (blood cultures, streptococcus and gonococcus, or examinations of pus), endocarditis (same means of diagnosis), and recurring fever (Ebstein's disease) due to lymphatic tuberculosis (tuberculin test). It is this type of fever that is seen in syphilis, and such a cause must be excluded. Murphy calls attention to the rapid rise and fall of the temperature in gall-stone cases.

Physical Diagnosis.—The following methods secure valuable facts, and must be employed in each case:

(1) Inspection. Fixation or restriction of the side.

(2) Palpation. To find out if pain, tenderness, swelling, or spasm is present.

(*a*) Simple. Tenderness at Mayo Robson's point, at the juncture of the outer one-third with the inner two-thirds of a line drawn from the tip of the ninth right cartilage to the umbilicus. Outline tumor. If inflamed gall-bladder, continuous with the liver; if distended, movable, pear-shaped, neck toward liver having tendency to disappear and bob up again.

(*b*) Bimanual. Fluctuation. Gall-stone rubbing.

(*c*) Method of Glenard.

(*d*) Eight fingers. Pressure upon four fingers placed over the part by four of the other hand, the fingers of the hand not pressing being relaxed. (Pottalschek.)

(*e*) Hooking the fingers under the rib in the region of the gall-bladder or pressing deeply in this region while the patient takes a full breath. (Murphy.)

(*f*) Deep prod with the closed fist (Jordan-Lloyd) over the gall-bladder excites pain if gall-stones are present.

(3) Percussion. Outline tumor, note if continuous with liver. An interval of resonance may exist between the liver and gall-bladder tumors. Auscultatory percussion is not conclusive. Note,

however, that percussion must be employed in both the upright and the recumbent postures to detect displacements of the liver.

4. Auscultation. Friction of perihepatitis; gall-stone crepitus (rare).

X-Rays.—Of some value in the hands of Beck. Usually considered doubtful.

Exploratory Puncture.—Not justifiable.

Laboratory Diagnosis.—(1) *The Blood.*—(*a*) *Leukocytosis.*—It is remarkable how few reports are made. Fuller information is required on this important point. It is present in acute cholecystitis and cholangitis, not typhoid. Its presence points to streptococcus infections, to pneumococcus infections, to bacillus coli infections, and excludes typhoid, malarial, and tuberculous infections. It often excludes amebic abscess, but does not exclude multiple abscess of the liver. If lymphocytes are in excess syphilis is suggested.

(*b*) *Iodophilia.*—Locke and Cabot found positive reaction in five out of seven cases of disease of the gall-ducts. Their studies, however, showed positive results in a very large percentage of cases in which suppuration prevailed. One can place about the same value upon it as upon leukocytosis.

(*c*) *Red Blood Count.*—This should be made repeatedly. In jaundice it falls rapidly, and the degree of reduction should guide one in the indications for operation.

(*d*) *Coagulation Time.*—A close watch of the blood should be made with Wright's tubes. The normal coagulation time is from two to four minutes. In jaundice the blood may coagulate so slowly that eight to ten minutes may elapse before clotting is completed.

(*e*) *Tuberculin Test.*—Jaundice may be due to enlarged lymphatic glands pressing on the ducts. Recurrent fever (Ebstein's) attends it. It may simulate gall-bladder infections. A tumor of the liver so situated as to resemble an enlarged gall-bladder may be tuberculous. The test mentioned will give the characteristic reaction.

(*f*) *The Urine.* (From studies by Dr. Edsall.)—The presence of bilirubin indicates obstruction. Observers have long tried to find some indications from urinary analyses to distinguish the various forms of hepatic disease and differentiate between liver and gall-duct disease. My colleague, Dr. Edsall, has been engaged in these studies, has studied some of my cases, and I am permitted to give

his results—which, however, like those of others who have done similar work, are either negative or extremely inconclusive. They may, nevertheless, have some interest. His report is as follows:

"(1) The neutral sulphur of the urine was early determined to be increased in certain disorders of the liver. I estimated the neutral sulphur in a series of cases of liver disease with and without jaundice, in connection with some other urinary constituents. The results were very irregular and seemed to be of absolutely no consequence in diagnosis, as was to have been expected from the general physiological stand-point. Others have reached the same conclusion.

"(2) Recently (Edsall states) I have published some results of the estimation of the 'readily eliminable' fraction of the urinary sulphur. There was some reason to believe that this might bear an important relation to liver disease. My results, together with those of Petry and Lang, apparently demonstrate that this fraction of the urinary sulphur is of no clinical importance of any kind.

"(3) I have also made a considerable series of estimations of the ratio between the total nitrogen and the ammonia nitrogen of the urine in liver disease and in other conditions. This was of some interest, because of the teaching, which has so long been prevalent, that most of the urea is formed (from ammonia) in the liver. My results agree with those of others that have recently mentioned this question, in showing that this ratio has no importance in the diagnosis of hepatic disease, and indeed, it is all but certain that much of the urea is formed elsewhere than in the liver, particularly when that organ is diseased.

"(4) Since Strauss referred to some suggestive results from the estimation of the volatile fatty acids of the urine, I have made a series of estimations of these. I have repeatedly found excessive amounts in liver disease; I have quite as often found them normal; and I have frequently found them much increased, when there was no other indication of even temporary disturbance of the liver. Comparatively little work has been done on the excretion of the volatile fatty acids in various diseases, and more observations would be of value. I do not believe, however, that they will offer much aid in the diagnosis of disorder of the liver.

"(5) Strauss has also recently reported some very interesting results from tests for alimentary levulosuria as a means of diag-

nosing disease of the liver; and Bruining and Ferrannini have had favorable results from the test. My own results in a small series of cases of cirrhosis have led me to believe that the test is of little or no practical value; for levulose did not appear in the urine of any of these cases in amounts sufficient to give a frank reduction of Fehling's solution or to cause distinct fermentation, and even the delicate Seliwanoff reaction was absent in half the cases. Moreover, this test is quite often positive in this slight degree in other conditions; then, too, it has no further experimental basis than the fact that Hans Sachs has shown that when the livers of frogs are removed, these animals do not assimilate levulose. It has never even been indicated that levulose is assimilated in frogs by the liver alone, or that this is so in any other animals. It is, indeed, extremely probable that other organs have a large share in this; hence, it is likely that liver disease has no constant or reliable relation to a disturbance in the assimilation of levulose.

"(6) I also began, some time ago, some work on the amidoacids of the urine in liver disease; but I have not carried this far. It was suggested by the recent work demonstrating the activity of autolysis in liver-substance, and by the well-known relation between acute yellow atrophy, etc., and the excretion of leucin and tyrosin. Von Jaksch has recently contributed a few observations related to this question, which are interesting. The matter would repay further study from a purely pathological stand-point, and perhaps from the clinical.

"(7) I have made numerous observations concerning urobilinuria, and can only agree with most other observers that it is, at most, suggestive of liver disease—and then, only when a number of other conditions can be reasonably excluded. The latter is a difficult task; and, further, urobilinuria is often absent in hepatic disease.

"It has been quite clearly established that urobilin is in most instances, at least, and in chief part, formed in the intestines by bacterial action. For this reason I have, with the aid of Dr. Fife and Dr. Wile, made a series of about 200 observations of the relations between urobilinuria and the excretion of various enterogenous decomposition products—a question that has hitherto received very little attention. A portion of these observations were made on cases that certainly had disease of the liver. The results indicate that

even in the absence of any demonstrable increase in blood-destruction, urobilinuria is not always dependent simply upon abnormal or increased intestinal decomposition-processes. Our results also lead me to think it probable that urobilinuria is commonly due to imperfect alteration of urobilin after its absorption from the digestive tract; and this imperfect alteration is probably due chiefly to disorder of the liver. This view is in accord with the growing opinion that excessive excretion of enterogenous decomposition-products often indicates disturbance in tissue-processes, rather than simply disturbance in the digestive tract. If this view can be demonstrated to be correct, it will indicate that urobilinuria is more directly related to disorder of the liver than is now generally taught; but more definite facts would be needed to give it greater diagnostic importance than it has at present.

"A number of observers have recently insisted that indicanuria is often an indication of disorder of the liver, rather than of the digestive tract. It is undoubtedly true that intense indicanuria, with or without urobilinuria, is often seen in liver disease, and frequently when any marked gastro-intestinal disturbance is absent. This is, however, an inconclusive fact, and one that offers no important aid in diagnosis.

"A fact that has interested me more than this is that I have repeatedly observed, in definite liver disease, and in chronic alcoholism when any distinctive signs of liver disease were, as yet, absent, that the distillate from the acidified urine persistently gave an intense phenol reaction (with bromin water), even when there were no noteworthy evidences of disturbance of the stomach or intestines. This intense phenol-reaction is common in infectious fevers and in marked gastro-intestinal disturbances. Except in such conditions, an intense reaction has, in my experience, been generally associated with disease of the liver, or with alcoholism or other conditions that gave rise to a suspicion of liver disease; and it has frequently been combined with a more or less marked urobilinuria. Herter has shown that liver-substance causes phenol to disappear, and it is also probable that the synthesis of phenol into conjugate sulphates takes place chiefly in the liver. These facts, the results that I have mentioned, and other reasons make it seem probable that phenol may bear some relation to liver disease that will prove to be of clinical importance. My observations concerning this point are,

as yet, very inconclusive. A mere excess of phenol in the urine is certainly not, of itself, in any way indicative of hepatic disease; but there are some features of the excretion of phenol that are, I believe, worthy of further attention in this connection."

(*g*) *Gastric Analyses.*—Such analyses carefully conducted will enable one to differentiate between liver and gastric conditions, and with the physical examinations will usually lead to the recognition of the gastroptosis and the dilatations which cause pain and simulate gall-bladder affections. It is well known that hyperacidity attends gall-stones. I found constant hyperacidity in two cases in young subjects who had symptoms of cholecystitis, but in whom I could not elicit the antecedent and attending phenomena of cholelithiasis. It occurred to me that as pancreatitis may be caused by hydrochloric acid, so cholecystitis might be induced if the acid could get into the ducts. Experiments conducted to determine this point by Drs. Yates and Pearce showed that in healthy subjects gastric juice could not be forced into the gall-bladder from the duodenum. The experiments were as follows:

(A) Two experiments—dead dog, pylorus ligated, duodenum below bile duct ligated. Bowel filled with eosin solution containing lamp-black in suspension. Forcible manual pressure for several minutes. Result negative. Duct not stained.

(B) Same experiment on human cadaver. Result negative.

(C) Live dog. Ligation of duodenum 20 cm. below pylorus. Injection of same fluid. Animal killed after five hours. Had not vomited, but was collapsed, powerless, and in dying condition when chloroformed. Examination—slight eosin staining at papilla but no extension along duct.

(D) Same experiment, but ligation below the ileocecal valve. Dog killed after sixteen hours. Result negative.

Notwithstanding these negative results it may be possible that under altered pathological conditions hydrochloric acid or gastric juice may get into the bile passages and cause inflammation.

Historical Diagnosis.—Reliance placed on the age, sex, habits, and other facts of the social history, and of the previous medical history, is essential in the diagnosis of secondary states. A glance at the secondary morbid states explains this statement.

Fallacy.—Attention must be called to the fact that the age of persons suffering from cholelithiasis, as determined at autopsy or

in the operating-room, is not that at which the disease began. The age thus recorded is the age when the secondary processes of calculi have taken place, that is, infection of the biliary passages in which gall-stones are present, and produce the symptoms which require treatment or cause death.

REPORT OF CASES

CASE I.—*Acute Cholecystitis. Absence of Gall-Stones; Severe Infection. Operation. Immediate Recovery.*

J. R., aged 49 years, married. Resided in Philadelphia.

"Always reckless with eating" as to time, character of food, and method of eating. Moderate in the use of beer and whiskey. Passed a renal calculus several years ago. Always inclined to constipation and subject to so-called bilious attacks, which were relieved by purgatives.

Nothing further of interest in the social, family, or previous medical history.

On April 4, 1902, he partook of a supper of indigestible food. April 5, 4 A.M., he was seized with severe pain in the epigastrium and the hepatic region. His physician found him with pain, tenderness over the region of the gall-bladder, a temperature of 101° F., and a pulse of 106. There was no nausea, vomiting, or movement of bowels.

Pain continued during the day, for which morphin was taken. Unable to secure a movement of the bowels throughout the day.

April 6. Fairly quiet night under influence of morphin. Temperature 100° F., pulse 104. Pain increasing; abdomen very tympanitic, greatest tenderness over the gall-bladder region. At 4 P.M., the temperature was 103° F., the pulse 112. The bowels moved for the first time, after high enema. At 5 P.M., a rigor; pain continued. At 8 P.M., the rigor was repeated; temperature 103.8° F., pulse 116.

April 7. Poor night; moderate collapse; temperature 101° F., pulse 116. Sweating; great tenderness over the epigastrium and the gall-bladder. A palpable tumor in gall-bladder region. Seen by Dr. Musser. Diagnosis, acute cholecystitis. At 12 M., temperature 101.8° F., pulse 108. Nausea. No bowel movement. Otherwise same. Urine contained bile pigment; otherwise normal. At 5 P.M., the temperature was 102° F., the pulse 116. Much pain.

Operation declined. At 11 P.M., the temperature was 101.9° F., the pulse 124, the respiration 22. Pain; patient nervous and restless.

April 8. Very restless during night; very little sleep. Six stools, thin, yellow. Nausea. Temperature 101.4° F., pulse 110. Some perspiration during the morning. Headache and backache. Pain and tympany. At 2 P.M., temperature 102° F., pulse 98. At 5 P.M., temperature 105° F., pulse 120. Leukocytosis of 12,000.

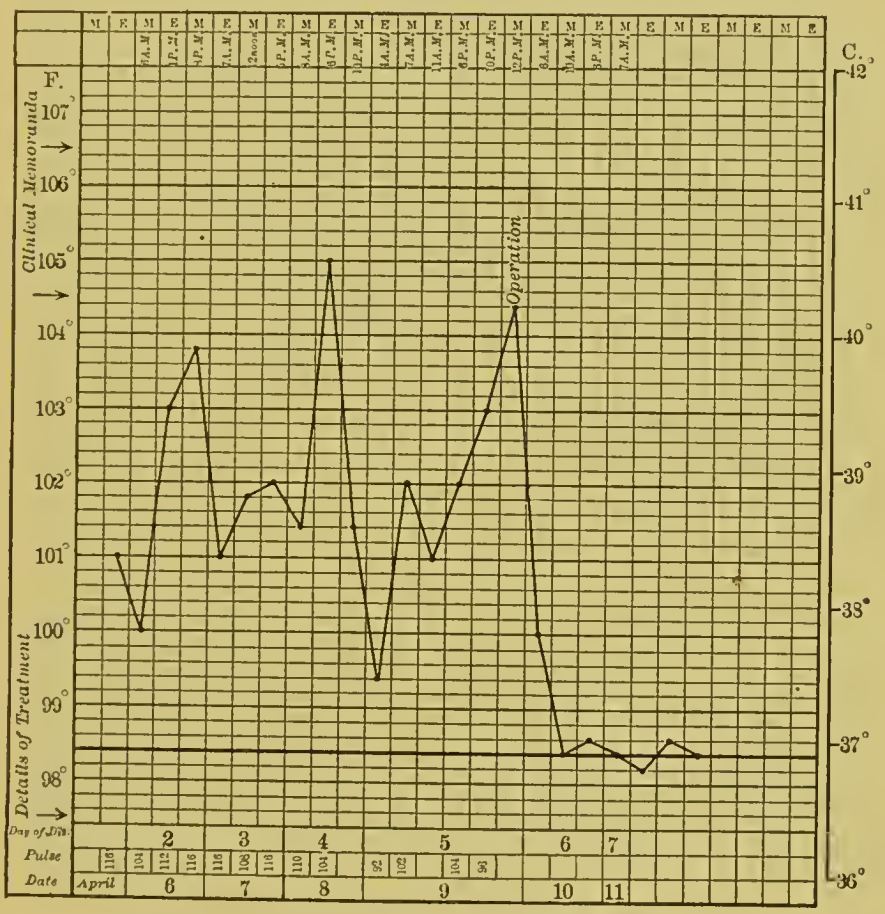


CHART I.—Acute cholecystitis. Operation. Recovery.

Backache and headache. Urine scanty and high colored. At 8 P.M., temperature 103.3° F., pulse 118. Thin, black stools during the evening. Local conditions the same.

April 9. At 4 A.M., temperature 99.4° F., pulse 92. At 8 A.M., temperature 102° F., pulse 102. Pain continues. Tumor distinct. At 12 M., temperature 101.2° F., pulse 96. Headache. At 3 P.M., local conditions more aggravated. Temperature 101.3° F.; pulse

100, respiration 28. Mild delirium; very short of breath. At 9 P.M., delirium increased along with other evidence of toxemia. Temperature 103° F., pulse 108, respiration 26. Throughout day several stools. Owing to manifest increase of toxemia and no relief to local symptoms, consent to operation, which Dr. Martin joined in advising, was given. Operation at 1 A.M. by Dr. Martin. Swollen, edematous gall-bladder surrounded by recent pericholecys-

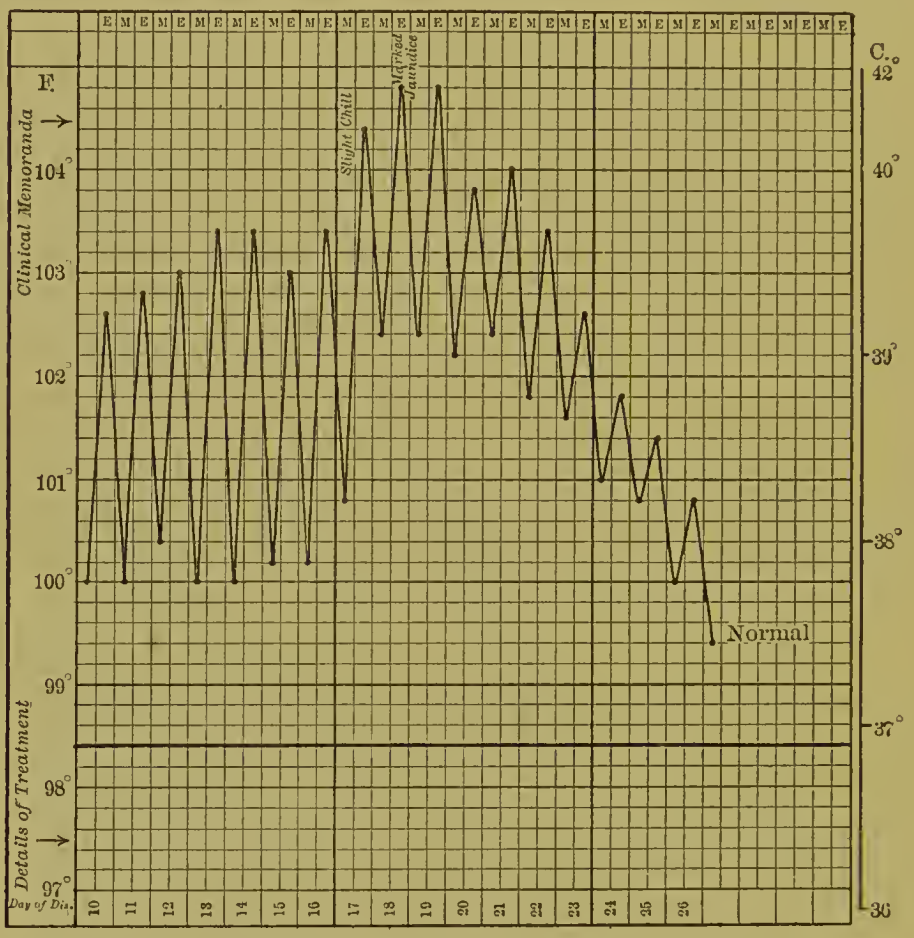


CHART II.—Cholangitis and cholecystitis in the course of typhoid fever.

titis. Distention due to enormous amount of seropurulent fluid. No gall-stones. Drainage. Immediate relief (see Chart I).

CASE II.—*Cholecystitis and Cholangitis in the Course of Typhoid Fever.*

February, 1903. In the third week, in the decline of the fever, of a moderately severe attack of typhoid fever, without any cause, increase of temperature developed, as shown in Chart II. At the end of four days I saw the patient in consultation. No unusual

symptoms. Slight nausea. Tongue furred. Marked, but not deep icterus. Tenderness of the gall-bladder, which was palpable. Liver not enlarged. Recovery in ten days.

CASE III.—*Typhoid Cholecystitis in Convalescence. Operation. Recovery.*

March, 1903. Patient, aged 55 years, married, female. Seen with Dr. Rehfuss. Typhoid fever of six weeks' duration. Four

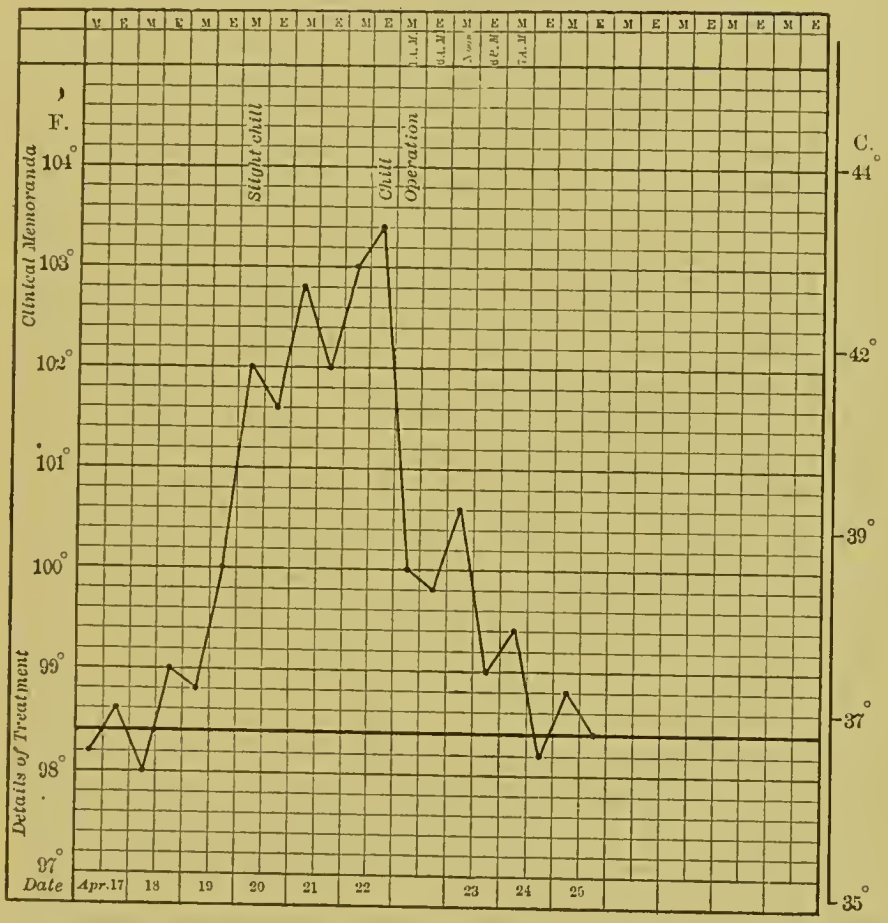


CHART III.—Cholecystitis in typhoid fever developing four days after the temperature reached normal. Operation. Recovery.

days after the temperature became normal she was seized with pain and vomiting. In the afternoon a chill occurred, and the temperature rose as shown on Chart III. The patient was seen by me on the third day, when tympany and tenderness in the region of the gall-bladder were present. The pulse was rapid and feeble. Constant vomiting. Bowels opened freely.

Physical examination indicated swelling, and the greatest ten-

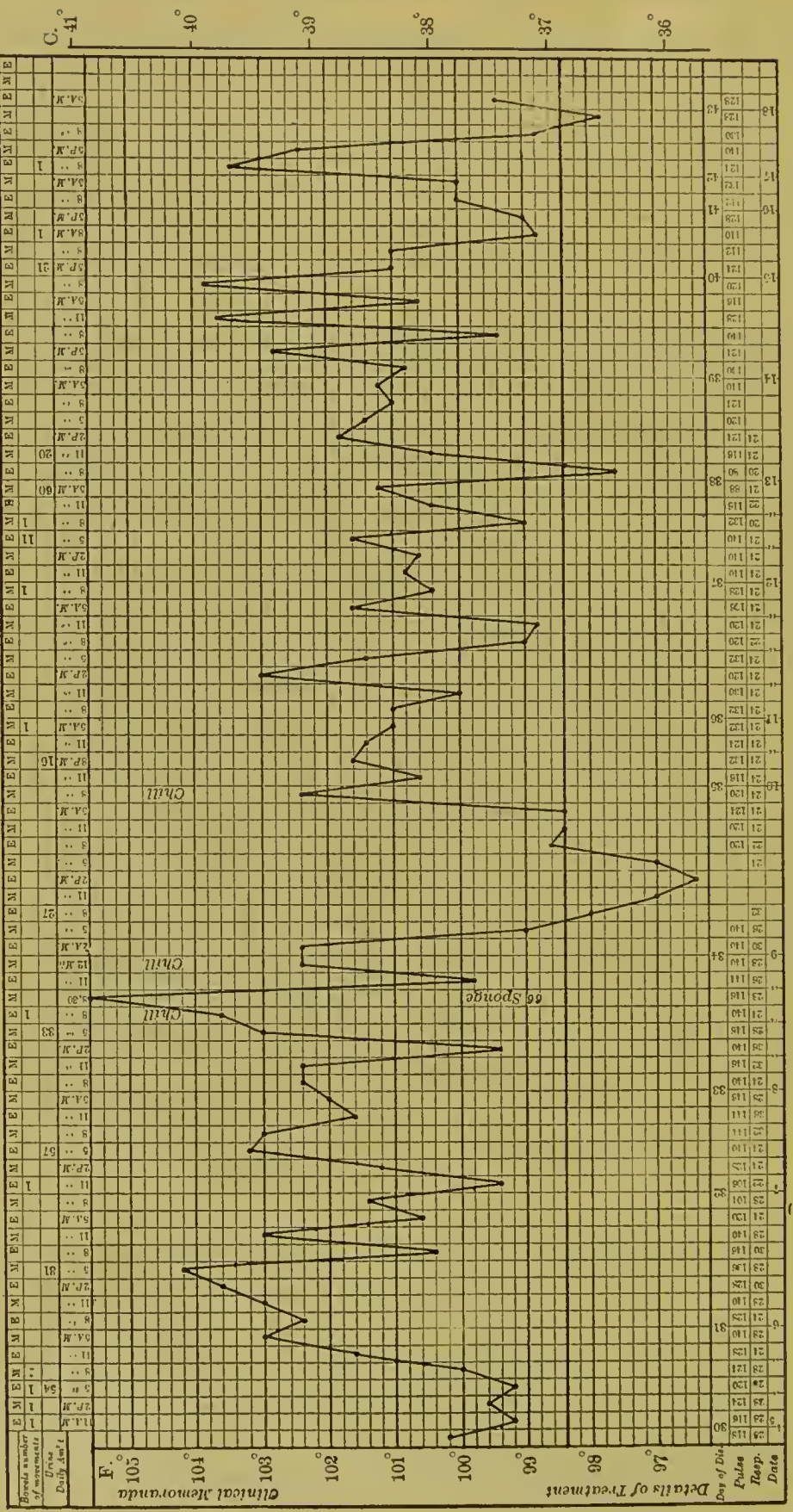


CHART IV.—Cholecystitis in typhoid fever developing forty-eight hours after the temperature reached normal. Death from acute hemorrhagic pancreatitis.

derness was between the end of the ninth rib and the umbilicus. Rigidity of right rectus marked. Appendicitis was excluded because of the history and the location of pain and rigidity. Perforation was excluded because of the time in the course of the disease, because of absence of shock and sudden pain. It was evident that localized peritonitis was advancing rapidly to general. Dr. Martin concurred in the diagnosis and operated four hours after my visit, when the pulse was 160, vomiting constant, and tympany great. Very extensive peritonitis about liver, gall-bladder, and in right upper quadrant was found; the gall-bladder was enlarged and distended. It was opened and drained, with prompt recovery, as indicated by the chart.

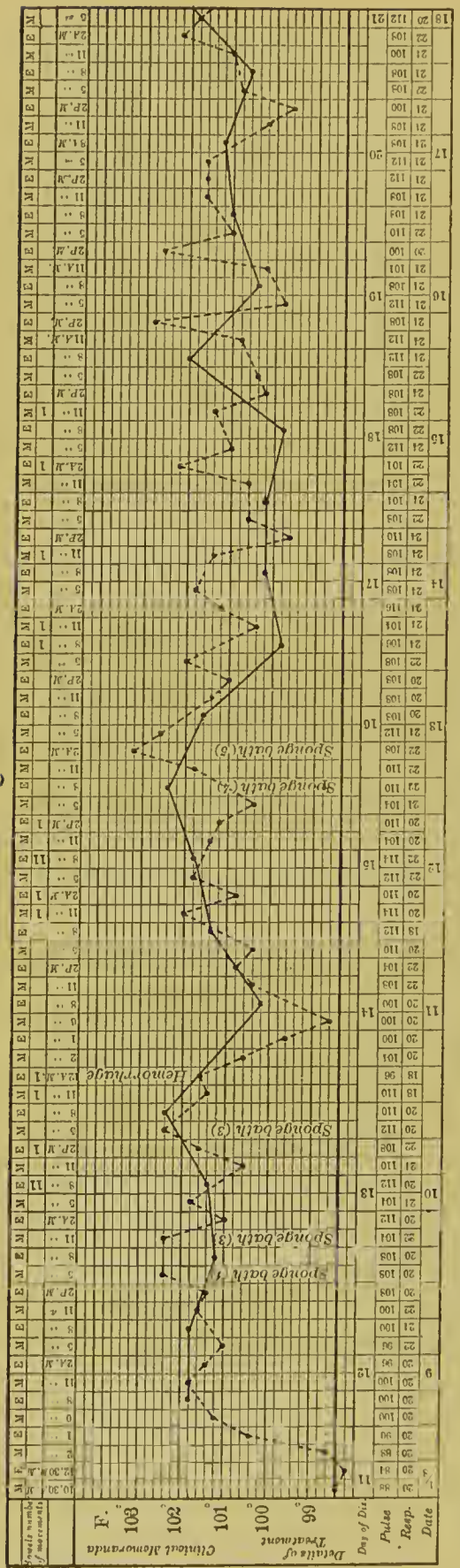
Case IV.—*Typhoid Fever. Cholecystitis. Acute Pancreatitis. Death.*

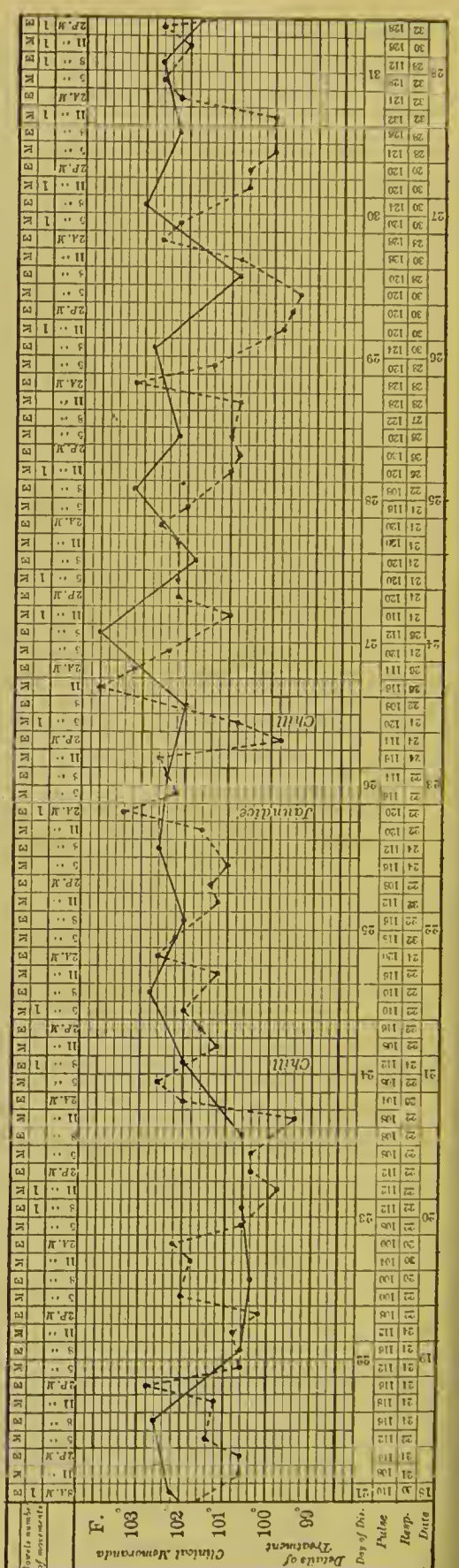
January, 1903. The patient was desperately ill with typhoid fever for six weeks. The fever did not subside except for 48 hours. A recurrence of fever with chills, as Chart IV indicates, was believed to be due to ugly bed-sores and suppuration from hypodermoclysis abscesses. There were no signs of abdominal disease. One day, after slight exertion, turning to drink, she died suddenly.

At the autopsy, suppurative cholecystitis without gall-stones, and acute hemorrhagic pancreatitis were found. The great interest in the case is in the sequence of pancreatitis upon typhoid cholecystitis.

Case V.—*Cholecystitis in the Course of Typhoid Fever. Recovery.*

The patient, a woman, aged 45 years, was seen in consultation with Dr. Taylor and Dr. Girvin, in 1901, on the twenty-fifth day of an attack of typhoid fever, on which day the temperature arose, and pain, rigidity, and tenderness in the gall-bladder region were found. A tumor the size of a large orange, movable with respiration, and on palpation tender, filled the region between the normal gall-bladder site and the umbilicus. The local symptoms were very severe for ten days, but gradually subsided, the temperature (see Chart V, pages 28 and 29) declining with the lessening of the abdominal symptoms.


Clinical Memoranda
Details of Treatment
Day of Dis.
Pulse
Resp.
Date
Sponge bath (3)
Sponge bath (2)
Sponge bath (3)
Sponge bath
Hemorrhage
Sponge bath (3)
Sponge bath (2)
Sponge bath (3)


Clinical Memoranda
Details of Treatment
Day of Dis.
Pulse
Resp.
Date
Jaundice
Chill
Chill

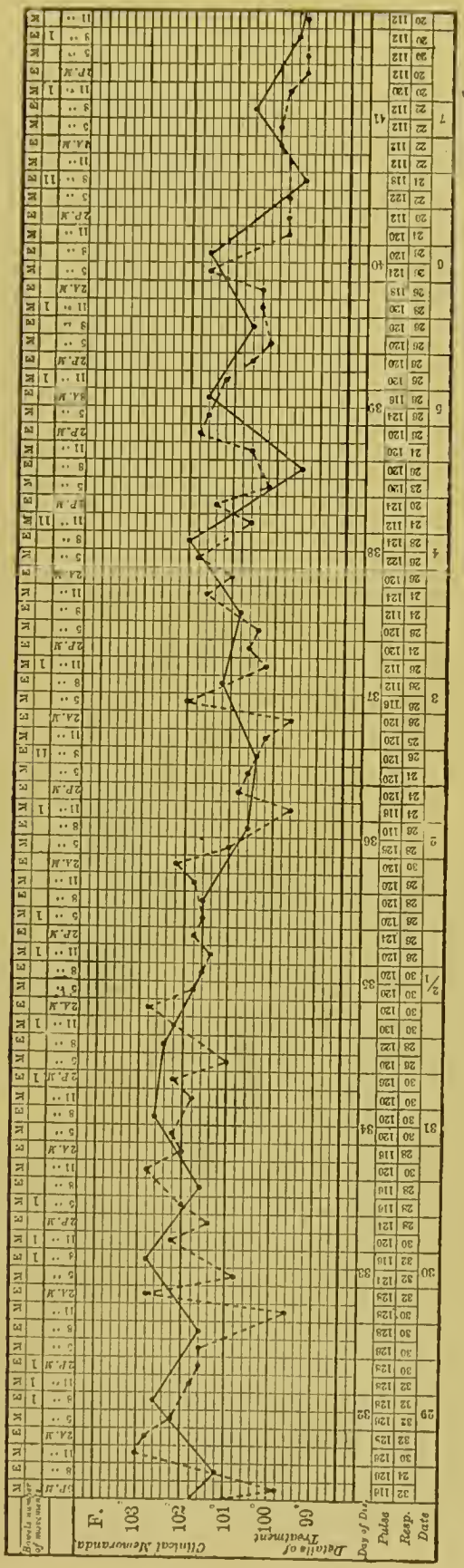


CHART V.—Cholecystitis in the course of typhoid fever.